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Edward T. Reichert

Extracted from the American Journal of the Medical Sciences for October, 1881.

CONTRIBUTIONS TO THE STUDY OF THE TOXICOLOGY OF CARDIAC DEPRESSANTS.

I. CARBOLIC ACID;

A SUMMARY OF FIFTY-SIX CASES OF POISONING, WITH A STUDY OF ITS PHYSIOLOGICAL ACTION.

BY

EDWARD T. REICHERT, M. D.,

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I. CARBOLIC ACID;

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Last June, while pursuing a series of experiments in connection with a paper on the dangerous properties of chlorinated, brominated, and iodated anæsthetics,[1] it occurred to the writer that all decided cardiac depressants were capable at times, like chloroform, of acting wholly out of proportion to the dose used, even under apparently similar circumstances, and several instances were cited of poisoning by aconite, apomorphine, and tartar emetic, in connection with the cases due to the above classes of anæsthetics, to illustrate this belief. Not feeling satisfied to allow so interesting a question, and one of such momentous importance to the therapeutist, to remain unsettled, a survey of the articles on the toxicological effects of cardiac depressants was made. But the absence of statistical papers was so noticeable, and the details given so inadequate, and oftentimes so inaccurate, that the urgent need was felt for a series of papers which would faithfully depict the poisonous phenomena and such other features as would seem to be of interest and value to the therapeutist and toxicologist.

I have selected carbolic acid as the earliest to be discussed, because the knowledge of its physiological action among the profession seems to be so very limited, and as no study of any large number of cases has yet been made—the deductions appearing in the several essays already published evidently being based on a consideration of but few cases.

[1] See page 50 of the preceding number of this Journal.

The following are the records of the cases, which have been very considerably condensed:—

Case I. Female, aged 43.—*Dose and preparation taken.* Amount (?); crude. —*Symptoms.* Insensibility; retching; breathing stertorous; pupils contracted; pulse intermittent.—*Treatment.* Chalk; 7 ounces of olive oil injected into the stomach by stomach pump, and subsequently an emetic was given.—*Result.* Death in six hours.

Autopsy and Remarks. Strong smell of carbolic acid detected; œsophagus dark gray colour, harsh to the touch, and the inner part friable and tearing readily. The mucous membrane of the stomach was in a similar condition. The intestines presented the appearance from a dark gray above to a bright red and congested appearance in the jejunum. Brain congested. Blood incoagulable, and remained fluid up to time of writing, which was five days. Brain contained ℥ij serous fluid. (Harrison, *Lancet*, 1868, ii. p. 133.)

Case II. Male, aged 64.—*Dose and preparation taken.* Rather over ℥j in ℥j of oil.—*Symptoms.* In ten minutes: the patient looked ill; flushed, perspiring, ineffectual attempt to vomit; eyes rolling wildly as if bewildered; mouth, tongue, and fauces whitened. (Mustard given, but stomach ineffectually evacuated; pump used and albumen given; castor oil injected into stomach.) In an hour: mental equilibrium restored; skin hot and perspiring; face flushed; pupils strongly contracted; pulse, rapid and soft; breathing somewhat laborious; voice harsh; deglutition difficult; thirst; retching; burning pain and tenderness at epigastrium. Acute gastritis supervened; features occasionally distorted by convulsive twitchings; hot, dry skin; temp. 102.5°, cold extremities; respiration shallow; pulse 140, small and hard; pupils contracted; intelligence perfect. Collapse ensued; imperceptible pulse; and cadaverous features.—*Treatment.* Emetics, albumen, castor oil, and stomach pump.—*Result.* Death in twelve hours from a failure of heart.

Autopsy and Remarks. The man had a compound comminuted fracture of the thigh, and in the above toxic condition bore manipulation with perfect equanimity, as if he was almost anæsthetized. (Anderson, *Lancet*, 1869, i. p. 179.)

Case III. Female, aged 44.—*Dose and preparation taken.* Nearly ℥j impure carbolic acid.—*Symptoms.* Dyspnœa; epigastric pain; restlessness; mouth and lips whitened; brown stain on the chin; pulse 140, feeble; difficulty in swallowing; stupor; breathing slow and stertorous; pulse exceedingly feeble; comatose. —*Treatment.* Zinc sulphate, olive oil; no vomiting ensued; stomach pump used, and warm water injected.—*Result.* Death in 50 minutes.

Autopsy and Remarks. Left lung greatly congested; right emphysematous; mucous membrane of both lungs congested; left ventricle of heart strongly contracted, right partially so; mucous membrane of mouth, œsophagus, and stomach converted into a soft white material; blood fluid, became light red on exposure. (Brabant, *Lancet*, 1873, i. p. 302; also in *Guy's Hosp. Rep.*, 1879.)

Case IV. Female, aged 7.—*Dose and preparation taken.* ℥ss diluted with ℥j glycerine and water.—*Symptoms.* Ten minutes—insensible; pulse almost imperceptible; pupils contracted; conjunctival reflex gone; temperature lowered; respiration catching and slowed; died comatose.—*Treatment.* Stomach pump, gruel and oil, enema of brandy; heat of body maintained.—*Result.* Death in 1 hour and 25 minutes.

Autopsy and Remarks. Right side of heart full of blood, which was dark and fluid; also larger vessels full of similar blood. Left ventricle contracted, and contained only two drachms of blood. No abnormality in lungs or abdominal viscera, except alimentary canal. The tongue, mouth, fauces, œsophagus, and stomach and upper part of duodenum unaffected by poison, but small intestine for three feet was whitened. Blood fluid; brain contained much venous blood; no effusion into the ventricles, which were quite dry; odour of carbolic acid detected in brain. (Rickards, *Lancet*, 1873, i. p. 876.)

Case V. Female, aged 20.—*Dose and preparation taken.* ℥iss impure undiluted acid.—*Symptoms.* Mouth and tongue whitened; pain in œsophagus and stomach; swallowed readily; hesitancy in speech in four or five minutes; stam-

mers; totters in walk; insensibility in eight minutes; unable to swallow; breathing stertorous; pupils unaffected; pulse frequent, small and intermittent; extremities warm up to the time of death.—*Treatment.* Six eggs given through stomach tube; water and magnesia; the contents of stomach drawn off; artificial respiration for half an hour with some benefit.—*Result.* Died in little more than an hour after taking the poison.

Autopsy and Remarks. Lips and nose blistered; sinus of dura mater gorged with dark tarry blood; bloodvessels on surface of brain full; lungs and heart healthy, the latter empty; mucous membrane of mouth whitened, and of œsophagus and stomach congested and peeled off with the slightest touch; mucous membrane of intestinal canal did not appear much changed; smell of carbolic acid in all the organs; green stains in the stomach. (Swain, *Lancet*, 1869, i. p. 395.)

Case VI. Male, adult.—*Dose and preparation taken.* Probably a mouthful of impure acid.—*Symptoms.* Probably ten minutes elapsed, then vomiting; comatose; contracted pupils; intermittent pulse; stertorous breathing; frothing at the mouth; livid appearance of eyelids, lips, and ears.—*Treatment.* Emetic by means of stomach-pump, but without success.—*Result.* Died in three-quarters of an hour.

Autopsy and Remarks. Tongue and fauces corroded. (Gerrard, *Lancet*, 1871. i. p. 672.)

Case VII. Female, aged 37.—*Dose and preparation taken.* f℥j of—acid 1–10 linseed oil—(nearly 50 grains).—*Symptoms.* Twelve minutes or more, sufficiently conscious to reply when spoken to sharply; skin moist; face dusky; pulse weak; pupils slightly dilated; unconsciousness; duskiness deepened; moisture of skin increased; pupils dilated; pulse quick, irregular, and scarcely perceptible; breathing regular and noisy. In three-quarters of an hour, coma complete; face livid; pupils dilated; body and extremities warm and moist; pulse imperceptible; soft palate and tongue relaxed; breathing slow and regular; reflex sensibility of conjunctiva was now altogether lost.—*Treatment.* Stomach washed with tepid water; the oil which came out did not smell of the acid; aqua ammoniæ (2 gtts. FFF.) injected into vein of forearm, and in a few seconds the radial pulse became perceptible. Repeated injection in one-quarter of an hour with benefit.—*Result.* Recovery. (Shaw, *Lancet*, 1875, ii. p. 451.)

Case VIII. Female, aged 29.—*Dose and preparation taken.* ℥ss (crude).—*Symptoms.* 5 minutes: collapse; cold; clammy; pulse 112, almost imperceptible; conjunctiva insensible; pupils dilated and immovable; respiratory movements almost imperceptible, and were 56 in a minute; froth from the nostrils; breathing stertorous; cardiac movements weaker and intermittent; pupils contracted somewhat; face livid.—*Treatment.* Olive oil given first with a spoon and then with pump; solution of sodium carbonate given; stomach emptied; brandy per rectum, and mixed with milk; sinapisms to legs.—*Result.* Died in forty-five minutes.

Autopsy and Remarks. Rigors strongly marked; dependent parts congested; skin corroded at angles of mouth; froth from inflamed nostrils; blood fluid, and smelt of carbolic acid; brain smelt of acid; ventricles of brain full of serous fluid, odour of carbolic acid detected; vessels full of fluid blood; trachea in a state of acute inflammatory congestion; lungs and bronchial tubes congested; heart pale and flabby, and contained fluid blood in all cavities; mouth and tongue blanched; mucous membrane of œsophagus white and rough, and the epithelium appeared replaced by false membrane which could be stripped off, leaving a red congested surface; cardiac end of stomach reddened, and in a state of acute inflammatory congestion, mucous membrane soft and smooth; bladder contained four ounces of urine, smelt of carbolic acid, but otherwise normal. (Packer, *Lancet*, 1878, ii. p. 511.)

Case IX. Male, aged 32.—*Dose and preparation taken.* 10 A. M., swallowed a quantity of a solution.—*Symptoms.* Immediately: nausea; cold sweats; stupor; unconsciousness (12.30, magnesia given); profound insensibility; noisy, tracheal rales in breathing; death apparently imminent. (Mustard poultices applied to the whole surface.) 5 P. M.: temperature risen nearly to normal; coma; complete relaxation of the limbs persisted; anæsthesia of skin and mucous

membrane of mouth, nose, pharynx, and bladder; complete loss of reflex excitability; cornea and conjunctiva insensible; pupils contracted; respiration (48) stertorous, with tracheal rales; pulse (128) small. He had passed no urine, but some which was drawn off was yellow, with a violet tint by reflected light, and which smelt of carbolic acid. On its surface were some oily drops. Some drawn blood was of a singular brown colour, and possessed the odour of the acid. The clot formed was soft and diffluent, and did not contract.—*Result.* Death in nine and a half hours.

Autopsy and Remarks. All parts smelt strongly of carbolic acid. No distinct lesions of the mucous membrane were found above the stomach. The membrane of the stomach was thickened, marked with black cauterized spots, and elsewhere deeply congested, and in one or two places were submucous hemorrhages; it was not ulcerated; kidneys deeply congested; some spots of effused blood under the capsule and in cortex; the tubuli were perfect; epithelium fatty and completely degenerated. Albumen and acid found in the urine. (Rendu and Patrouillard, *Jour. de Pharm. et de Chem.*, Dec. 1871; *New Remedies*, April, 1872, p. 341.)

Case X. Male, aged 60.—*Dose and preparation taken.* Not more than ʒj. (?) —*Symptoms.* Frothing at mouth; countenance turgid; quite speechless.—*Result.* Died in a few minutes of shock and depression.

Autopsy and Remarks. Body discoloured; superficial veins engorged; mouth and throat whitened; faint odour of carbolic acid; veins of scalp gorged with dark blood; brain free from any sign of congestion; but little fluid in cerebral ventricles, merely moisture; lungs full of dark blood, but normal; heart pale, fatty, quite empty; stomach contracted and coats thickened; contained about an ounce of turbid, pinkish fluid, smelling strongly of carbolic acid and giving a strong reaction; mucous membrane whitened on tops of furrows but reddish in furrows; œsophagus slightly affected; no trace of severe local effects in intestines, which were reddened along superficial surface. The appearance of the mucous membrane extraordinary and characteristic; cardiac end of stomach principally affected. (Barlow, *Lancet*, 1869, ii. p. 404.)

Case XI. Female, aged 60.—*Dose and preparation taken.* ʒvj (?) Calvert's used in XI., XII., and XIII.; applied acid to surface of body; had itch.—*Symptoms.* In a few moments smarting pain and headache; giddiness, approaching insensibility. Twenty-five minutes: prostration; spasmodic breathing; total insensibility; surface of body hard and dry; occasional spasm of diaphragm; pupils unaffected and responded to light. Continued unconscious for four hours, the breathing was laboured and slow.—*Treatment.* Acid washed from skin by tepid water and soft soap; brandy with ammonia and ether, some was swallowed. —*Result.* Died in four (?) hours.

Autopsy and Remarks. Small patches of sloughy skin about the body. (Machin, *British Med. Journ.*, 1868, i. p. 220.)

Case XII. Female, aged 23.—*Dose and preparation taken.* (See Case XI.) Applied acid to surface of body.—*Symptoms.* (See, also, Case XI.) Unconscious for five hours; consciousness gradually returned; attempts at vomiting; mustard given; but little of stomach contents were evacuated; retching considerable; strong odour of carbolic acid in expiration; respiration lost spasmodic character, quick and irregular; continued pain in head and throat. In the morning epileptiform convulsions came on; pain in head better, but throat unrelieved; symptoms of congestion of lungs; gradually sank; conscious until the last. Patient said to be an epileptic, and to have had convulsions when application was made.—*Treatment.* Emetic of mustard given with but little effect; strong coffee given with benefit; leeches to temples in course of morning; alkaline mixture of potassium chlorate and chlorine given, but symptoms of congestion of lungs came on and patient collapsed.—*Result.* Died in about forty hours.

Autopsy and Remarks. Small patches of sloughy skin about the body. (Machin, *British Med. Journ.*, 1868, i. p. 220.)

Case XIII. Female, aged 68.—*Dose and preparation taken.* (See Case XI.); applied the acid to surface of body; had itch.—*Symptoms.* (See, also, Case XI.) Rallied in about four hours; states that as soon as dressing was used, she felt giddy-drunk; head felt constricted by band; unconsciousness; no vomiting in this case;

pulse (80) regular and feeble; skin harsh, dry, puckered; no vesication in any of the three cases (XI., XII., XIII). Skin thrown off often in scaly patches.—*Treatment.* (As in Case XI.)—*Result.* Recovered in twenty-one days. (Machin, *British Med. Journ.*, 1868, i. p. 223.)

Case XIV. Female, aged 51.—*Dose and preparation taken.* A case of excision of elbow-joint. Dressed with lotion, one part to fifty.—*Symptoms, etc.* Dec. 2, seventy-two hours: "shivering fit;" tongue rapidly fouled; pulse 100–120, weak and irregular; skin cold and clammy; very uncontrollable vomiting; face pinched and anxious; spirits depressed. Fifth day, rapidly sinking; pulse 130–140, and thready; tongue has a brown coat; cerebration clear; urine scanty and loaded with lithates, but not dark or carbonaceous. The carbolic lotion was the same day changed and flaxseed used instead, and in the course of forty hours pus was present in poultices, and the constitutional irritability removed. Improving up to Dec. 15, when lotion was again employed (same strength). Thirty-six hours: vomiting, and same symptoms as before. The discharge, which was purulent on the 15th, became scanty and rusty on the 17th; poultices again substituted on the 18th.—Poultices used up to the 27th, when lotion was applied with a similar result as above; wound was now nearly healed. The lotion was discontinued and warm water used.—*Result.* Recovered.

Remarks. Nothing was found in this case to be able to check the vomiting caused by the carbolic acid. (Lightfoot, *British Med Journ.*, 1870, i. p. 331.)

Case XV. *Dose and preparation taken.* One part to five of linseed oil, used as a dressing for bedsores in typhus fever.—*Symptoms.* Two days dressed with lint soaked with the carbolic acid and oil. After the second dressing severe vomiting; urine became very dark. On discontinuing carbolized oil, the symptoms ceased, and urine became natural. No signs of blood corpuscles in the urine could be detected with the microscope, only carbonaceous particles.—*Result.* Recovered. (Wilks, quoted by Lightfoot, *Brit. Med. Journ.*, i., 1870, p. 332; also *Guy's Hosp. Reports.*)

Case XVI. Male, adult.—*Dose and preparation taken.* Carbolic enema.—*Symptoms.* During administration fit occurred; stertorous breathing.—*Result.* Death in twenty minutes. (Worcester Infirmary, quoted by Lightfoot; Ibid.)

Case XVII. Female, aged 1 year.—Had extensive ulceration of gluteal region and labia, following from sitting on block on which some of the disinfectant, ℥j to Oj of water, had been thrown two days before.—*Symptoms*, etc. The next morning after sitting on the block the blistering had commenced and ulceration rapidly followed. Used fullers' earth and French chalk with comforting effect. On fifth day used lotion of lead-water with glycerine, several sloughs formed and separated; child became weaker from exhaustion and shock; some signs of internal inflammation; usual remedies used.—*Result.* Died on tenth day.

Autopsy and Remarks. Rectum inflamed; patches of inflammation in intestine; right pleura much inflamed. Child had been perfectly healthy before. (Sandwell. *British Med. Journ.*, ii., 1870, p. 382.)

Case XVIII. Male, aged 47.—*Dose and preparation taken.* ℥j or ℥ij (?) (crude, black and oily).—*Symptoms.* Shortly afterward found insensible; smelt of carbolic acid; speechless; cannot walk; unconscious; stertorous breathing; puffing of lips at expiration; heat of body natural; pulse disturbed (84–106) and intermitting; emetic had no effect; lips, gums, and tongue whitened; no muscular movements in three or four hours; eyelids closed; pupils contracted to a pin's point; at six and one-half hours opened eyes and moved the extremities; after this pupils expanded; began to speak; asked for cold water, but never regained complete consciousness. Then laborious breathing occurred; difficult muco-purulent expectoration; breathing easier and coma lessened; severe pain in back; respiration 46–48. Eight and one-half hours: passed a quantity of very dark-coloured urine, which smelt of carbolic acid. Afternoon, attempted to pass water, but unsuccessfully; brow, face, and hands covered with a clammy perspiration; collapse.—*Treatment.* Castor oil, olive oil, and emetic; stomach-pump (would not work); ammonium carbonate and brandy, but to no purpose.—*Result.* Died in thirteen and one-half hours.

Autopsy and Remarks. Lividity of back and front of neck and chest; face sallow; pupils natural; watery froth and yellowish fluid at mouth and nostrils,

which smelt of carbolic acid; clot in longitudinal sinus; dura mater and arachnoid congested; blood smells of carbolic acid; serum found in ventricles and subarachnoid; gray matter of brain of pink hue; intense congestion of vessels on under surface of pons and medulla. Mucous membrane of mouth, throat, and gullet whitened, sodden, soft, and easily detached; ℥ij thick brownish pulp in stomach; great curvature covered with hard, reddish, and elevated points the size of shot, without any marked congestion; similar points on other portions of stomach, tending to follow the course of the vessels; stomach otherwise normal, perhaps more vascular. Portions of intestine of slate colour; containing brownish pulpy substance; heart full of dark firmly clotted blood on both sides, right side ℥iv, left ℥ij; ℥iv whitish urine in bladder; universal smell of carbolic acid; blood everywhere firmly clotted. (Ogston, *Brit. Med. Journ.*, 1871, i., p. 116.)

Case XIX. Male, adult. *Dose and preparation taken.* Exposed for three hours to fumes of strong carbolic acid.—*Symptoms.* Violent convulsions with trismus; quite comatose; lividity of face; stertorous breathing; extremities and surface cold; pulse scarcely perceptible; warm bath was given; in forty minutes convulsions ceased; sensibility partly restored; face and neck assume a natural hue; symptoms improved. Six hours later, passed urine, which was more acid than normal, but no odour of carbolic acid; complained of giddiness; no pain in head, but pain in face and eyes, with taste of carbolic acid in mouth and throat, and gastric irritation.—*Treatment.* Wrapped in blankets; hot water to feet; cold applications to head, and mustard to nucha; aperient mixture, bismuth subnitrate, dilute hydrocyanic acid (P. L.), a gargle of potassium chlorate and myrrh, and a liniment of belladonna with soap liniment to neck and face; warm baths.—*Result.* Recovered. (Nuthank, *Brit. Med. Journ.*, 1872, ii., p. 579.)

Case XX. Male, aged 7.—*Dose and preparation taken.* Probably a mouthful of the impure carbolic acid.—*Symptoms.* Stertorous breathing; complete stupor; total muscular relaxation; anæsthesia; pulse feeble and rapid (160); temperature lowered; pupils contracted; salivation; power of swallowing lost; failure of respiration and pulse; collapse.—*Result.* Died in about seven hours after symptoms were first observed.

Autopsy and Remarks. Post-mortem in twenty-four hours: rigor mortis present, but not to any marked degree; cerebral sinuses distended by dark fluid blood and some soft coagula; cerebral vessels in a similar condition; little fluid in subarachnoid space and ventricles; faint odour of acid detected, also in thorax, but intensified; venous congestion of thorax and abdomen; blood dark and fluid; lungs congested, œdematous, and emphysematous; heart empty, left ventricle contracted, right one flaccid, also empty; no coagula in great vessels; mucous membrane of mouth, throat, and œsophagus white, sodden, congested, but not detached; stomach same character; contained some injection, but no capillary hemorrhage; liver, spleen, and kidneys hyperæmic; bladder contained eight ounces urine of an olive-green tint, peculiar mixed odour, contained no albumen; carbolic acid detected in urine with chemical tests, and also in viscera. (Ferrier, *Brit. Med. Journ*, 1873, i. p. 167.)

Case XXI. Male, aged 36. *Dose and preparation taken.* Probably ℥j of undiluted acid.—*Symptoms.* Five minutes: mortal agony; lividity; protrusion of eyeballs; continued subdued cry of "wild and fear-inspiring tone," which was broken by gasping respiration and inefficient attempts to vomit; condition followed almost immediately by profound insensibility. From the first the pulse was very rapid and feeble; respiration short and infrequent; pupils unaffected.—*Treatment.* Olive oil poured in the mouth; warm water was injected in stomach and withdrawn, impregnated with carbolic acid; second injection of olive oil; difficulty of introducing pump experienced.—*Result.* Died in thirty minutes, apparently asphyxiated.

Autopsy and Remarks. Superficial veins, especially, extremely distended with blood; lips and chin excoriated; dura mater congested with dark venous blood; arachnoid at vertex thickened with gelatinous matter; brain healthy; lungs emphysematous in front, passive congestion posteriorly; pericardium contained ℥j serum; heart healthy, all cavities full of blood, which was dark and perfectly fluid,

with no coagula; mucous membrane of larynx, trachea, and subdivisions highly congested, granular, and softened; and air-tubes contained small quantities of olive oil and carbolic acid; tongue shrunken, pale, indurated; mucous membrane of œsophagus white, glistening, softened, peeling off as if scaled; mucous membrane of stomach softened and eroded, and with muscular coats reduced to a state of pulp, parts above orifices sustained most injury, and were of a dull, deep slate colour; small intestines similarly affected. (Hearder, *Brit. Med. Journ.*, 1878, i. p. 584.)

Case XXII. Male, adult.—*Dose and preparation taken.* Two or three drachms in a mixture.—*Symptoms.* Immediately fell insensible and convulsed, and in eighteen minutes extremities were cold; pulse scarcely perceptible and irregular; breathing stertorous; consciousness lost; trismus.—*Treatment.* Stomach evacuated; bled to Oj.—*Result.* Recovered on eleventh day. (Hearder, *loc. cit.*)

Case XXIII. Male, aged 72.—The patient was suffering with a suppurating stump.—*Dose and preparation taken.* Sucked carbolic acid from a sponge which had been placed under the bed as a disinfectant.—*Symptoms.* Semi-comatose; pulse (98) feeble; respiration 48; olive oil and castor oil given, which was swallowed very slowly, also zinc sulphate and tartar emetic; no vomiting; pulse and respiration better; coma.—*Treatment.* Olive and castor oil; ℈j zinc sulphate; two grains tartar emetic.—*Result.* Death in about four hours.

Autopsy and Remarks. Lips, chin, right side of face, right breast and hand coloured brown, and a strong odour of carbolic acid; dura mater and arachnoid very much congested; brain substance healthy. Mucous membrane of mouth and tongue white and softened, and of larynx thickened; of pharynx and œsophagus pale, thickened, soft, and corroded; of stomach soft, corroded, congested, and also white. Intestines softened, and part corroded; odour of carbolic acid detected in the cæcum. (Biddle, *Brit. Med. Journ.*, 1873, i. p. 611.)

Case XXIV. Female, aged 43.—*Dose and preparation taken.* ℥j impure carbolic acid.—*Symptoms.* Five minutes: insensible; face blanched and perspiring; pupils contracted; pulse 100, feeble and irregular; respiration stertorous, breath smelt of carbolic acid; slight lividity of lips and tips of fingers. Rapidly grew worse, and became much swollen before death.—*Treatment.* Emetic and stomach-pump; the patient could not swallow the emetic, and the pump only brought out a small quantity of contents which smelt strongly of carbolic acid; difficulty of introducing pump experienced.—*Result.* Died in one and a half hours.

Autopsy and Remarks. Angles of mouth and skin discoloured and shrivelled; interior of mouth whitened; tongue dry and chippy; mucous membrane of œsophagus dry, shrivelled, and brownish; heart feeble, both ventricles were empty; mucous membrane of stomach readily peeled from walls, white patches over it, and whole slightly inflamed; mucous membrane of duodenum similarly affected, but less so; dura mater natural; arachnoid thickened and opaque; brain apparently healthy. (Sutton, *Med. Times and Gaz.*, 1868, i. p. 456.)

Case XXV. Male, aged 19. The patient was suffering with enteric fever, and the acid given by mistake.—*Dose and preparation taken.* ℥ss impure carbolic acid.—*Symptoms.* Unable to swallow; almost pulseless; difficult respiration; lividity and a mottled appearance; stools black-brown; urine dark brown or blackish; collapse.—*Treatment.* Brandy given; nutrient enemata of beef-tea and brandy, and iced milk by mouth.—*Result.* Died in two days. (Wiltshire, *Med. Times and Gaz.*, 1870, ii. p. 474.)

Case XXVI. Male, aged 65.—*Dose and preparation taken.* ℥ss–℥j (?) impure acid.—*Symptoms.* Insensible; stertorous breathing; mouth and throat filled with mucus; stains of acid on chin; pupil contracted; pulse 40–50, and laboured; life nearly extinct; respiration irregular; heart-beats inaudible.—*Result.* Died in fifty minutes.

Autopsy and Remarks. General aspect as if dead by asphyxia; brownish lines from angles of mouth. Blood dark, fluid. No coagula, except in lungs. Mucous membrane of mouth and œsophagus whitened, firm, congested, hard, and tough. Stomach contained ℥iv of thick, turbid fluid; smells of carbolic acid. Mucous

membrane white, shrivelled, in granular masses, and easily scraped off; rugæ hard. Larynx, trachea, and bronchi filled with transparent mucus, streaked with blood. Heart flabby, and slightly fatty; left side contained little blood; right empty. Viscera congested. Arachnoid cavity contained ʒx fluid. (Jeffreys and Hainsworth, *Med. Times and Gaz.*, 1871, i. p. 423.)

Case XXVII. Female, aged 7.—*Dose and preparation taken.* Probably a mouthful, drunk from a demijohn of it used for disinfectant purposes.—*Symptoms.* Lips white, shining, and puckered; odour of carbolic acid detected; face of a leaden hue; cold sweat; eyes fixed; pupils slightly *dilated;* extremities cold; complete insensibility. Inability to induce emesis. Some improvement after use of stomach pump. Consciousness returned one and half hours after poisoning; heart feeble; pulse at wrist not countable. Pain in epigastrium. In three and three-quarter hours, respiration 40; pulse small and could not be counted. Pupils *contracted;* extremities cold. Three and three-quarter hours later, pulse 144; respiration 36; temperature 98°; less jactitation; drinks milk; "feels sick;" pupils widely *dilated;* relaxation of bowels. Twenty-third hour, face pale; respirations somewhat stertorous; slight convulsions; eyelids partly closed; semi-conscious; asks for milk constantly; lividity less; pupils *dilated;* extremities cold; respirations 50; relaxation of bowels. In fifteen minutes, unconsciousness; increased stertor; died five minutes later.—*Treatment.* Egg beaten up and poured down throat with a pump, three minutes after poison was taken, and shortly after albumen was given in same way. Later, a mixture of olive and castor oils, and afterwards wine of ipecac, were given. Stomach pump brought out albuminous coagula, which smelt of carbolic acid.—*Result.* Died in twenty-four hours.

Remarks. This patient was suffering with ascites. (Woodman, *Med. Times and Gaz.*, 1875, i. p. 421.)

Case XXVIII. Female, aged 40.—*Dose and preparation taken.* ʒiv crude carbolic acid, which was taken at 9 o'clock in the evening.—*Symptoms.* In twenty minutes, uneasiness; collapsed; blanched; no vomiting or convulsions; respiration stertorous; pupils contracted; pulse hardly perceptible, not rapid. Washed out the stomach. Two hours later, condition better, but remained unconscious. Midnight, semi-conscious; pupils contracted; pulse quick and feeble; surface cold; convulsive twitchings; vomited bloody and oily matter. Some return of consciousness; able to swallow brandy and milk, but vomited. Late that day the evacuations of the bowels were black, and afterwards dark brown. Urine smoky colour and aromatic odour; no albumen. Epigastric tenderness. Dark-brown stain on upper lip, mouth, and chin. For a couple of days later she remained in a dangerous condition, and the urine remained smoky; she then improved slowly.—*Treatment.* Stomach pump; mustard to calves of legs and heart; stomach thoroughly washed; Oj olive oil injected. First day, milk and brandy, barley water, milk and lime-water, and ice.—*Result.* Recovery; left the hospital on the 17th, having taken poison before midnight on the 15th. (Davidson, *Med. Times and Gaz.*, 1875, ii. p. 597.)

Case XXIX. Male, aged 57.—*Dose and preparation taken.* Amount (?) eighty per cent. acid.—*Symptoms.* Carried into hospital insensible, and emetics given. Vomited freely. Tongue and mouth were quite white; large white patch from mouth to chin; sore throat during the night, but well otherwise. Olive oil applied locally, and a mixture of mucilage given, ʒj, every four hours, internally. Next day, sore throat; urine dark, clear, faintly acid, no albumen, sp. gr. 1044. Carbolic acid found in urine. Second day, sore throat; fauces covered with white patches; tongue corrugated; bowels open; appetite good; left hospital in the afternoon —*Treatment.* 25 grains zinc sulphate effectual; olive oil externally; mist. mucil. ʒj every four hours.—*Result.* Recovered; discharged second day. (Forster, Guy's Hosp., *Med. Press and Circular*, 1878, ii. p. 42.)

Case XXX. Male, adult.—*Dose and preparation taken.* About ʒss Calvert's No. 5.—*Symptoms.* "Sudden and fatal sedation of nervous centres." Probably died without suffering.—*Treatment.* Emetics given, but produced no effect.—*Result.* Died in ten or fifteen minutes. (Hill, *Rich. and Louis. Med. Journ.;* quoted in *N. Y. Med. Record*, 1873, viii. p. 383.)

Case XXXI. Male, adult.—*Dose and preparation taken.* ℥ss impure.—*Symptoms.* Suffered no uneasiness, except burning pain in throat for a few days. He had been an opium eater, and took some olive oil as soon as he found out his mistake.—*Treatment.* Olive oil.—*Result.* Recovered. (Outerbridge, *N. Y. Med. Record*, 1873, viii. p. 517.)

Case XXXII. Male, aged 2½.—*Dose and preparation taken.* ʒj pure; equal to Calvert No. 2.—*Symptoms.* Immediately child ran about the room crying; then instantly became unconscious; lividity; eyes staring; respiration stertorous; foaming at the mouth and nose; pupils dilated, but responding to light; twitching of muscles of the extremities; capillary circulation slow. Vomiting excited to a slight degree by titillating the fauces; quantity of tenacious, partially coagulated mucus expelled at each attempt at vomiting. Vomited mucus mixed with blood. Temperature raised; cough croupy. Throat whitened and shrivelled. Urine dark olive-green colour, with odour of the acid. Stools greenish, thin; became dark and smoky after standing some days.—*Treatment.* Olive oil; white of egg; vomited; milk and lime-water; bismuth and chalk every three hours.—*Result.* Recovered (Dusau, *N. Y. Med. Record*, 1878, xiii. p. 289.)

Case XXXIII. Male, aged 5.—*Dose and preparation taken.* Cavity of open abscess injected with diluted acid, according to Callender, of London.—*Symptoms.* Radial pulse absent; skin pale, cold, and damp; respiration short, hurried, and over 100. (Brandy given hypodermically); pupils contracted. Belladonna given and repeated, pupils then became dilated; pulse perceptible; respiration 100, reduced to 70.—*Treatment.* Hypodermic injections of brandy; belladonna.—*Result.* Recovered. (Post, *N. Y. Med. Record*, 1879, xv. p. 378.)

Case XXXIV. Aged 2.—*Dose and preparation taken.* ʒj of mixture of equal parts carbolic acid and olive oil taken at eight o'clock.—*Symptoms.* Five minutes, pain in bowels; soon became insensible. Ten minutes, partially comatose; pulse 120; difficult breathing; congestion of lungs; paralysis of muscles of pharynx. 8.30, insensible; left pupil slightly dilated, but sensitive; extremities cold. 8.45, left iris more dilated, less sensitive; breathing apparently improving under ammonia. 9, paralysis more marked; breathing becoming difficult, artificial respiration necessary. 9.20, pupils more sensitive to light; congestion of lungs becoming less. 9.30, pulse 140; breathing about normal; extremities warm. 10, improving. 12.30, vomiting some; oil given. Next day better; complains of pain in head.—*Treatment.* Carbonate of ammonia; castor oil.—*Result.* Recovered in a few days. (Ghent, *Canada Lancet*, 1875, i. p. 67.)

Case XXXV. Female, aged 50.—*Dose and preparation taken.* ʒij; woman in delicate health.—*Symptoms.* Did not complain of much burning sensation; gave sodium bicarbonate and olive oil, followed by extract of ipecac. General debility; violent burning sensation from fauces to stomach; pulse slow, full, regular; semi-comatose; can scarcely move her limbs; relaxed; with difficulty swallowed oil. Pulse unchanged. Forty minutes after taking poison, apomorphia given, followed by emesis; vomit smelt of carbolic acid. Violent efforts at emesis every twenty minutes for one and a half hours. No sinking or flagging of pulse; no cold extremities. Urine copious, smoky colour, smells strongly of carbolic acid; continued copious for twelve hours longer, and gradually improved; bowels relaxed. Five and a half hours after poisoning, pulse 96, full and regular; temperature 101°. Lay in a critical condition for two days; then greatly improved. —*Treatment.* Sodium bicarbonate, olive oil, enema of oil, apomorphia (about half a grain) hypodermically, mucilage, milk, and water.—*Result.* Recovered. (Semple, *Virginia Med. Monthly*, May, 1877, iv. p. 138.)

Case XXXVI. Female, aged 39. Suffering with carcinoma uteri; the neck being nearly destroyed, leaving the body of uterus open.—*Dose and preparation taken.* Applied, locally, cotton-wool pessaries saturated with carbolic acid, glycerine, and oil, the carbolic being in the proportion of one part to eight of liquid. —*Symptoms.* This treatment was continued from Nov. 17th to Jan. 7th, when she suddenly became insensible; convulsions; inability to void urine. Suppression of urine continued until the 11th, when some water was discovered in the bed. 17th, another convulsion; fatal collapse. For several days before death, anasarca.—*Treatment.* Dry-cupped loins, hot fomentations, salines, ten-

minim doses tr. digitalis.—*Result.* Died. (Edwards, *Practitioner*, 1869, iii. p. 324.)

Case XXXVII. Female, aged 41. Suffering with cancer.—*Dose and preparation taken.* Injection ordered of "one part of carbolic acid, glycerine ℥j. and ℥xx water." This was used *ter in die* for some days; then "applied daily a cotton-wool pessary saturated with carbolic acid, tannin, and glycerine, the strength being one of acid to sixteen parts of water."—*Symptoms.* Commenced treatment Sept. 6th. Sept. 27th, hiccough; sickness; prussic acid and salines given, with a blister to epigastrium. Oct. 2d, rigors; extremities cold. 3d, partially unconscious; face œdematous; legs much swollen; no water passed. 4th, no better; delirium at night; no water passed, and bladder emptied by passing a catheter. 5th, hemorrhage (uterine); anasarca; and at times, insensibility. 10 P. M. drew off ℥ij bloody urine. Oct. 8th, decidedly worse. 10th, little more urine passed. 11th, died comatose.—*Result.* Died. (Edwards, *Practitioner*, 1869, iii. p. 324.)

Case XXXVIII. Male, aged 40.—*Dose and preparation taken.* Probably a whiskeyglassful of the impure acid.—*Symptoms.* Pallor; coldness of surface; insensibility, and coma; respiration slow and laboured; pupils fixed and insensible to light; no erosion or mark on face or lips. Stomach pump brought up a quantity of dark fluid. Next day passed dark, almost black, urine; no carbolic acid or albumen in secretion. Pulse frequent and weak.—*Treatment.* Turpentine enema, sinapisms, stomach pump, ammonia to nostrils, opium, and morphia.—*Result.* Recovered. (Warren, *Irish Hosp. Gaz.*, 1875, iii. p. 17.)

Case XXXIX. Female, aged 33.—*Dose and preparation taken.* ℥ss liquefied carbolic acid.—*Symptoms.* Immediately "felt mad;" "fought the air." Fifteen minutes, uneasiness. 4 o'clock, uneasiness; extremities somewhat flexed and rigid; trismus; pupils contracted; pulse feeble; respiration attended with "rattles." 7 A. M. pulse extremely weak; surface cold. 12.30, severe burning in stomach; sore throat; painful deglutition; headache; no thirst; temperature 98°; pulse 90 and weak. 7 P. M. delirious at times; nausea, but no vomiting; hurried respiration.—*Treatment.* Morphia, brandy, beef-tea, milk, eggs, etc.—*Result.* Recovered. (Walker, *Boston Med. and Surg. Journ.*, 1879, p. 797.)

Case XL. Female, aged 35.—*Dose and preparation taken.* Scarcely less than ℥viij.—*Result.* Died very soon.

Autopsy and Remarks. Mucous membrane of œsophagus, stomach, and small intestine thickened and corrugated, and of a bluish-white colour. Action of poison ceased abruptly fifty inches from pylorus. Œsophagus at point of stomach so much contracted that the point of a probe was passed with difficulty. Blood fluid, very dark; cavity of heart moderately full. Viscera and urine exhaled the odour of the acid. Mode of dying seems to be *syncope* or *shock*. (Way, *Trans. Path. Soc.*, 1873, xxiv. p. 93.)

Case XLI. Aged 21 months.—*Dose and preparation taken.* ℥j crude carbolic acid.—*Symptoms.* Ten minutes, motionless; insensible, but recovered in a little time. Pupils contracted, insensible to light; conjunctiva insensible; pulse 120 and very weak; cold and clammy; lividity of lips; respiration impeded; strong tarry odour of breath; vomited frothy liquid. Unable to swallow emetic. Breathing grew worse; (artificial respiration used); lividity worse; (tracheotomy, which enabled the child to breathe easier). Four hours, respiration 80; anæsthesia; contraction of pupil. Child vomited later.—*Treatment.* Unable to introduce emetic into the stomach, as œsophageal walls seemed spasmodically contracted; artificial respiration; tracheotomy performed.—*Result.* Died in ten hours.

Autopsy. Twelve hours after death. Brown discoloration of skin about mouth, which is dry and shrunken. Bronchi contained a brown-red liquid, which choked tubes and smelt of acid. Mouth whitened; dense epithelium loosely connected; œsophagus the same. White line terminated abruptly at stomach. Stomach, at anterior part, lower curvature, and in isolated patches on anterior and posterior surfaces, greatly changed. Change consisted of red-based patches with red borders, mostly limited to rugæ. Surface diphtheritic; walls of the jejunum natural. (Taylor, *Guy's Hosp. Rep.*, 1868, xiii. p. 233.)

Case XLII. Male, aged 50.—*Dose and preparation taken.* Injection into piles (internal) of a "liquid composed partly of carbolic acid." Had used on two occasions before with success, five weeks before the present one.—*Symptoms.* 11 A. M. Felt at once "uncomfortable." 4 o'clock, had a chill, with nervous distress. Passed a bad night: sleeplessness; nervous distress; insupportable giddiness and strange feeling about the head. 6 A. M. treatment for carbolic-acid poisoning commenced. Afternoon, "nervous chill, although warm;" weak, irregular pulse; giddiness, and impending convulsions. Giddiness continued for three days.—*Treatment.* "Eliminative character, with the bromides in addition."—*Result.* Recovered. (Wright, *Cinn. Lan. and Clin.*, Aug. 1878, p. 68; vol. i., New Series.)

Case XLIII. Female, aged 22.—*Dose and preparation taken.* ℥ss Calvert (No. 4). Patient just recovering from a second attack of relapsing fever. Took poison at 4.15 A. M. on September 17th.—*Symptoms.* 5 minutes, complete unconsciousness; conjunctival reflex gone; pupils normal and insensitive to light; surface livid, clammy; perspiration, copious; extremities cold; respiration husky, with considerable mucous rattle; occasional moaning. Pulse shaky, feeble; no whiteness or unusual appearance about the lips. Power of deglutition gone. 5.30, pulse improved. 7.30, able to swallow. 8.30, greatly improved; pulse 108, soft and feeble: lividity gone; body warm, but clammy. 12.30, passed green-black urine; vomiting; stools light-coloured and offensive; looseness of bowels. 18th, urine more clear and light. 19th, symptoms double basic pneumonia. Pulse ranged between 108 and 130 until Oct. 13th, when she suddenly became worse and died at 9 A. M.—*Treatment.* ℥j brandy and milk enema; with stomach tube, gave in course of half hour Oj and ℥iss brandy. Fomentations to abdomen; hot bottles to feet.—*Result.* Died.

Autopsy and Remarks. Autopsy: The woman had double pneumonia, which the author attributes to the effects of the acid. (Tennent, *Glasgow Med. Journ.*, 1870–1, p. 74–78.)

Case XLIV. Male, adult. Acute eczema.—*Dose and preparation taken.* One part of carbolic acid to four of lard applied on lint to arms and thighs, and covered with oiled silk.—*Symptoms.* In an hour and a half "profound coma; pupils firmly contracted; breathing stertorous; pulse weak, quick, flickering; inability to swallow.—*Result.* (?) (*Canada Med. Journ.*, 1871, vii. p. 8; quoted by Tennent.)

Case XLV. Male, aged 32.—*Dose and preparation taken.* ℥ss in the oily state.—*Symptoms.* Five minutes, insensible; face livid; bathed in profuse, clammy perspiration; eyes open and turned up; pupils contracted and do not respond to light; mouth open, filled with frothy mucus; respiration stertorous; pulse imperceptible; heart sounds scarcely audible; forced to swallow raw eggs and ℥vj mustard water—vomited; could not pass tube on account of stricture of œsophagus.—*Treatment.* Raw eggs, mustard water, galvanic current, and artificial respiration; by stomach tube large quantities of soap, lime-water, olive oil, flaxseed mucilage, and fluids were given; also counter-irritation.—*Result.* Died in one and a quarter hours.

Autopsy and Remarks. Eighteen hours after death: Rigor well marked; blood dark and fluid, smelt strongly of carbolic acid; venous system greatly congested; membranes of brain congested; sinuses all filled with dark blood; mouth, pharynx, and œsophagus whitened; stomach contained about ℥viij chocolate-coloured fluid, mucous membrane chocolate coloured, roughened, and charred, and in some places completely destroyed, exposing the muscular layer, and everywhere readily detached; destruction mostly near cardiac pouch; the pylorus thickened, and velvety to the touch; discoloration and destruction continued through the duodenum, fading along small intestines; whole alimentary tract highly congested; heart flabby and empty; lungs highly congested; bronchi contained frothy mucus; kidneys and liver congested. (Houston, *Med. and Surg. Rep.*, 1870, xxii. p. 32.)

Case XLVI. Male, aged 23.—*Dose and preparation taken.* Probably a good swallow of the impure—"not likely more than an ounce."—*Symptoms.* Two minutes: insensible, pulseless; pupils dilated; face pallid and pinched; respira-

tion gasping; involuntary discharge of urine; respiratory interval prolonged: heart ceased; then the respirations.—*Result.* Died in about three minutes, not more.

Autopsy and Remarks. Stomach moderately distended, intensely congested; externally being of a dark venous hue; contained Oj of whitish-coloured liquid, smelling strongly of carbolic acid; mucous membrane has a white film, and beneath this was congestion and a chocolate coloration: the membrane was strongly corrugated, thick, higher and more rigid than normal, especially marked at cardiac extremity, and along the greater curvature, and in lower end of œsophagus. Scalp and meninges congested, and vessels filled with dark fluid blood. (Taylor, *Phil. Med. Times*, 1873, ii. p. 284.)

Case XLVII. Male, aged 48.—*Dose and preparation taken.* Gr. v of crystals in ℥ij water.—*Symptoms.* Immediately experienced burning in the throat and stomach; vertigo; nausea, with retching, but could not vomit; paleness; hyperæmia of fauces; pulse 90; cold and nervous (gave lime-water, tartar emetic, and ipecac); no vomiting; nausea and vertigo; giddiness continued for eight hours, when emesis occurred, which relieved his nausea; but the vertigo trouble persisted until the following day.—*Treatment.* Lime-water, tartar emetic, and ipecac.—*Result.* Recovered. (Winslow, *Phil. Med. Times*, 1874, iv. p. 817.)

Case XLVIII. Aged two years.—*Dose and preparation taken.* ℥ij Calvert, No. 4.—*Symptoms.* Immediately screamed; drank ℥iv lime-water, ℥j olive oil, then insensibility supervened; ten minutes, profound coma; tactile sensibility totally abolished; surface pallid; copious perspiration; limbs flaccid; eyelids closed, bluish; pinched expression; frothy saliva. Pulse (160) soft, feeble, irregular; respiration (70) laboured, irregular, jerking, stertorous, and hissing; mucous membrane of tongue and fauces white, with patches of marginal streaks of capillary injection. Pupils widely dilated, and do not respond to light. Oil placed in pharynx immediately rejected, attended with suffocative symptoms. Apomorphine given occasionally; shudder, followed by slight clonic convulsions; prolonged spasm of glottis causing deep cyanosis. Any attempt to clear the mouth, or to give anything, excited dangerous spasm of glottis and general convulsions. A prick of a pin would cause the same thing. In the course of a couple of hours consciousness returned, and he took considerable powdered chalk and sugar, white of egg, iced port wine, and milk. Died with meningitic symptoms.—*Treatment.* Lime-water and olive oil; apomorphine.—*Result.* Died in twenty hours. (Winslow, *Ibid.*)

Case XLIX. Female, aged 20.—*Dose and preparation taken.* A hundred and forty-five grains in glycerine as an enema for ascarides.—*Symptoms.* She rapidly became convulsed, delirious, and finally nearly or quite insensible: surface cold and moist; pulse weak and flickering; pupils contracted; breathing stertorous.—*Treatment.* Injection of milk, and rectum thoroughly washed; ammonia, camphor, and diffusible stimulants given by the stomach.—*Result.* Recovered. (Pinkham, *Med. and Surg. Rep.*, xix. 1868, p. 492.)

Case L. Female, aged 29.—*Dose and preparation taken.* Half teacupful of carbolic lotion.—*Symptoms.* Vomiting soon began, accompanied with drowsiness; pupils natural. No drugs given. Free from symptoms next day. Urine a deep brown colour.—*Result.* Recovered. (*St. George's Hosp. Reports*, 1879, p. 25.)

Case LI. Male, adult.—*Dose and preparation taken.* Drank from a jug in mistake for whiskey.—*Symptoms.* In three minutes unconsciousness; no noticeable symptoms outside of the feeble, fluttering pulse, and frothing at the mouth. —*Result.* Died.

Autopsy and Remarks. Œsophagus contracted, and shows well-marked longitudinal folds; surface smooth, and of a peculiar bright, slightly yellowish-white colour, "closely approaching that of freshly-pulled molasses candy;" stomach strongly contracted, and summits of rugæ marked with white lines, and in interstices were found granular detritus. (Harris, *Boston Med. and Surg Journ.*, 1880, i. p. 494.)

Case LII. Female, aged 40.—*Dose and preparation taken.* "One or more swallows, ninety-five per cent. acid"—"at least ℥ij entered stomach."—*Symptoms.* Five minutes: comatose; respiration stertorous; pupils contracted and insensible; profuse perspiration. (Tartar emetic failed to produce emesis.)—

Treatment. Tartar emetic, lime-water, and olive oil freely.—*Result.* Died in three hours. (Park, *Chicago Med. Gaz.*, 1880, i. p. 228.)

Case LIII. Male, adult.—*Dose and preparation taken.* Lotion to necrosed tibia.—*Symptoms.* Pulse very feeble and flickering; respiration difficult; pale and anxious; feet and hands cold. (Wineglassful of brandy.) In three or four hours patient felt better, but a little nervous. (Opiate.) Urine next day smelt strongly of carbolic acid.—*Result.* Recovered. (White, *N. Y. Med. Gaz.*, 1871, p. 274.)

Case LIV. Male, aged 20.—*Dose and preparation taken.* ℥j pure fluid carbolic acid.—*Symptoms.* First swallowed milk, raw eggs, glycerine, and mustard; then inability to swallow. Half hour after poisoning, comatose condition; uneasiness, except when aroused; short, laborious, somewhat stertorous respiration; coolness, lividity, perspiration; feeble, almost imperceptible pulse.—*Treatment.* Forced down milk through tube. Pumped out the contents of stomach, and forced in milk and sweet oil, and pumped out again, and repeated.—*Result.* Recovered.

Remarks. Vomited when recovered from coma. Felt as well as usual next morning, except a slight burning in the stomach. (Glisan, *Amer. Journal of Med. Sciences*, 1880, lxxx. p. 452.)

Case LV. Female, aged 13.—*Dose and preparation taken.* ℥ij pure carbolic acid. Water immediately given; then emetic, with no effect.—*Symptoms.* In three minutes unconsciousness; seven minutes "radial pulsation gave way;" eight to twelve minutes tonic spasms, mainly of flexors; breathing slightly stertorous. —*Treatment.* Enema of brandy and milk, and stimulating applications.—*Result.* Died in nineteen minutes.

Autopsy and Remarks. Considerable corrosion of mouth and throat. (Sigler, *Louisville Med. News*, 1880, ix.–x. p. 294.)

Case LVI. Male, aged 24. Had herpes on right side, extending from nipple to axilla, about five inches square.—*Dose and preparation taken.* Painted the herpetic part with a saturated solution of the acid.—*Symptoms.* Twenty minutes, faint, dizziness, weakness in legs, and symptoms of collapse. Symptoms lasted for half an hour.—*Result.* Recovered. (Paul, *Phil. Med. Times*, 1880, x. p. 404.)

Besides these cases, there have been reported seven others in English journals to which I have not had access: Cleaver (*Liverpool Med. Surg. Rep.*, 1871, v. 45); Wood (*Ibid.*, p. 110); Maloney (*Australia Med. and Surg. Journ.*, 1872, xvii. p. 73–77); Allen (*Ibid.*, 1880, ii. p. 116); Peters, (*Indian Med. Gaz.*, 1868, viii. p. 423); Sheddon (*Glasgow Med. Journ.*, 1872, v. p. 266); Woodham (*Virginia Clin. Record*, 1871–2, i. p. 272). There are also to be found a number in journals of other languages.

Superficially reviewing the general symptoms of carbolic acid poisoning presented by the above cases, we find that the poison is absorbed with great rapidity, and the evidences of its toxic properties are soon manifested. In the course of a few minutes they become so rapidly aggravated that the system is profoundly affected; and, indeed, at least two cases offer good illustrations of its acting with the same deadliness and frightful rapidity as have been noted in cases of poisoning from the more virulent hydrocyanic acid. In cases which have been under observation from the time of the ingestion of the poison, symptoms have been observed to appear almost immediately, which indicate that the acid finds its way into the circulation as soon as it comes in contact with an absorbent surface, whether it be the skin, stomach, rectum, pulmonary acini, or wounds.

The first symptom generally appreciated is a burning pain in the parts of the body with which the acid has come in contact, provided the acid has been of sufficient concentration; in some cases, however, no pain is experienced and

even a diminution of sensibility may be present. Disorders of cerebration are then likely to follow, as evinced by a confused condition of thought, difficulty of speech, depression, restlessness and bewilderment, vertigo, delirium. Insensibility is one of the most frequent, earliest, and constant of the phenomena, but occasionally it does not make its appearance until late. Nausea and vomiting may be present, but this does not occur in more than about twenty per cent. of the cases. Uncontrollable vomiting occurred in one case, while in another it was very severe, and in a third was moderate. Even drugs seem to have but little power to bring about emesis, for in about twenty per cent. of the cases in which the different emetics were given, vomiting occurred only in about one-half of them. The pulse and respiration, like the nervous system, are seriously affected. The former in a large percentage of the cases becomes feeble, frequent, and intermittent, but in very exceptional instances was slow and weak. The respiration was soon affected, and in more than half of the cases, where it was particularly noted, was stertorous to a greater or less degree, but generally well marked; and in rare instances is described as being dyspnœic, slow, husky, or hurried. Occasionally the face is flushed, but more often a paleness is present. The skin is exceptionally hot and dry, and in a larger portion of cases is cold and clammy—especially noticeable in the extremities; and in a single case a hot skin was followed by coldness.

Lividity, more or less marked, is a frequently noted symptom; salivation was also observed. The temperature was stated to have risen above the normal in three cases, while in two it was lowered, and in a sixth case was natural. Frothing at the mouth and nose occurred in about ten per cent. of the cases, and deglutition was difficult in five and impossible in ten. In four the odour of the acid was detected in the exhalations. In ten cases the urine was noted as being coloured greenish, blackish, or brownish. Relaxation of the bowels was also occasionally noticed, and the stools in several instances were described as being brownish, blackish, or greenish. The pupils were variously affected, but in over half of the cases in which they were specially examined were found to be contracted; in less than a fifth they were dilated; in four were unaffected; in two dilatation was followed by contraction; and in another, there was first dilatation, then contraction, and ultimately dilatation. Convulsive movements occurred in fourteen cases; in one they were violent; in three were mere twitchings, and in a similar number trismus was present. In one case epileptiform convulsions were present, but this patient was stated to have been an epileptic, and in another case impending convulsions were recorded; sometimes they are only facial. Anæsthesia was also recorded in about fifteen per cent. of the cases, but was undoubtedly present in the vast majority where insensibility was recorded to signify this condition together with a loss of consciousness. Protrusion of the eyeballs, pain in the head or back, hiccough, muscular weakness, tremors, thirst, harshness of

voice, and involuntary micturition were also exceptionally noted. Symptoms of pneumonia, pulmonary congestion, or of meningitis, or mucopurulent expectoration, were observed in six cases.

Pain in the mouth, œsophagus, and stomach, frothing at the mouth or nose, insensibility, difficult or impossible deglutition, a cold and clammy skin, stertorous respiration, a frequent, small, and intermittent pulse, contracted pupils, anæsthesia, and a brownish, blackish, or greenish urine are the most constant and characteristic symptoms, and the latter is probably pathognomonic.

Experiments made on the lower animals yield results in accordance with the symptoms produced in man. According to Lemaire (*De l'Acide Phénique*, Paris, 1865), Neumann (*Archiv f. Derm. u. Syph.*, i. 1869, p. 425), Labbée (*Archiv. Gén. de Méd.*, 1871, p. 45), Stone (*Phila. Med. Times*, ix., 1878–9, p. 616), and Salkowski (*Pflüger's Archiv*, Bd. v., 1872), toxic doses in frogs produce paralysis of the posterior extremities followed by paralysis anteriorily, and of the whole muscular system. Lemaire states, however, that when frogs are placed in aqueous solution the anterior extremities are first affected. On the mouse and rat, Kempster (*Amer. Jour. of Med. Sciences*, July, 1868, p. 37) found that when they were confined to a jar and a sponge suspended a short distance from the bottom impregnated with the acid and the animal compelled to inhale the vapour, well-marked symptoms of poisoning rapidly occurred. The mouse in five minutes staggered as if intoxicated and then grew better. These paroxysms recurred several times during an hour and a half, when the animal became convulsed and died. A rat, in less than a minute, appeared sleepy, and as if intoxicated, muscular weakness was also apparent. In three-quarters of an hour it failed to notice sounds, and shortly after, to all appearances, was in a state of profound anæsthesia. Tremors, convulsions, and death followed. Two other experiments on rats confirm these results. In pigeons, according to Husemann (*Schmidt's Jahr.*, Bd. clv. p. 274), the same characteristic results occur as in mammals. Salkowski (*loc. cit.*), Neumann (*loc. cit.*), Lemaire (*loc. cit.*), and Husemann (*loc. cit.*) found in rabbits and dogs, tremors, muscular weakness, slowing of the respiration, dyspnœa, salivation, dilated pupils, diminished sensibility, convulsions, and death. Before convulsions rabbits lie on their sides kicking in mid-air. Temperature is diminished, and, according to Husemann, hæmaturia and albuminuria are occasional phenomena. A very curious fact was also noticed in frogs that, after awhile, violent convulsions were induced by very slight irritation; and at times when the posterior extremities were paralyzed, the anterior remained in a state of exalted reflex excitability (Stone, and Bert and Jolyet).

The *nervous system* is profoundly affected, and the train of symptoms following the action of the acid on this portion of the economy is decidedly well marked. *Convulsions* in the lower animals are more prominent phe-

nomena than in man, and are almost constant symptoms. Husemann states them to be one of the characteristic phenomena; but in man in less than about one-third only of the above cases were such movements present, and in only one were they at all violent. The convulsions are generally clonic, but may assume a decidedly tetanic character. That they are not peripheral seems proven by the experiments of Salkowski, Labbée, and Stone, for these observers found that they did not occur in the extremities which had been deprived of their central nervous connections by severing the nerves going to them; although they did occur when the limb was protected from the poison by ligaturing the bloodvessels. It is, therefore, certain that they must be central in their origin. Salkowski, Bert and Jolyet (*Gaz. Méd. de Paris*, 1872, xxvii. p. 188, etc.), and Stone (*loc. cit.*) found them occur in animals with cut cords, and hence they concluded that the convulsions were of spinal origin. These results, however, are not in accord with those of Labbée and Haynes (*Phila. Med. Times*, 1874, iv. p. 407), who state that convulsions did not occur in animals thus operated upon. From the disparity in these records it would seem that these phenomena are both spinal and cerebral in their origin, or else the former investigators did not completely sever the cord in the animals operated upon. Labbée removed the optic lobes and cerebri of animals, and found the convulsions still occurred when the basal ganglia were irritated, so that if the convulsions are exclusively cerebral they must, undoubtedly, if these results are accurate, be due to an action on the centres at the base of the brain; and the presumption that they are probably cerebral seems to be strengthened by the fact of their clonic character, yet the occurrence of a tetanoid-paralysant condition in frogs after they are profoundly under the influence of the poison suggests that these convulsions are dependent upon a similar action to that which produces like phenomena in the poisoning by atropine, apomorphine, and strychnia,[1] and hence, must be of spinal origin. Labbée does not give the details of his experiments; and Haynes used such large doses that it seems readily explainable why he observed convulsions anteriorly only; for, as above noted, the anterior extremities may be in a state of exalted reflex excitability while the posterior are paralyzed; and, moreover, when paralysis occurs it attacks the posterior extremities first—indeed, large doses may immediately cause general paralysis. When we consider the excessive doses used by Haynes (ʒij–ʒiij liquefied acid), it seems clear that the reason why he did not get convulsions posteriorly was because these large doses brought about an immediate condition of paralysis in the posterior extremities, with an attendant condition of hyper-excitability anteriorly. If, as before noted, we have a coexistent condition of paralysis and convulsions as has been described by several observers, it must be clear that if convulsions did not occur in the

[1] Reichert, Phila. Med. Times, 1881, p. 711.

posterior extremities, after section of the cord, unless the section was made during the convulsive stage, a negative result would be of little value as proof that convulsions were cerebral; while a positive result, such as obtained by at least three experimenters, would be very weighty in proving their spinal origin, and especially so, since the experiment was performed many times by a single investigator with the same result. It is extremely improbable that in every one of these experiments in which section of the cord was made it was incomplete.

Reflex action, according to Labbée (*loc. cit.*), is not lost, but he gives no details of experiments. Stone, in a very careful investigation of this function, obtained very decided results, and found that reflex action in frogs was primarily diminished and secondarily increased,[1] and that large doses may cause a decided exaltation. He also found that after section of the cord in the dorsal region hyperexcitability in the posterior extremities was not induced, nor was the diminished reflex activity present in the early part of the experiment as before; he therefore concludes that the diminished reflex irritability, as well as the increase, was due to a stimulation and ultimately to a depression of Setschenow's inhibitory centres in the base of the brain. This, however, appears to be inaccurate because after section of the cord convulsions occurred which were induced by slight stimuli, and consequently there must be some other reason for this condition of reflex excitability besides a depression of these centres, and it is probable that this exaltation is due, in part at least, to a depression of spinal reflex inhibitory centres which are similar in their function to Setschenow's. Late in the experiment, when the sensory function appeared to be highly exalted, the motor function was decidedly diminished, and Stone thinks that these phenomena were, like those of strychnia poisoning, due, as Kölliker suggested, to the excessive strain on the motor nerves; yet, this can scarcely be true if his own statement, as well as those of Salkowski (*loc. cit.*) and Hoppe-Seyler (*Pflüger's Archiv*, 1872, Bd. v.) be correct: that the motor and sensory nerves and muscles are not materially affected; and then again it is exceedingly improbable that the depression of the motor nerves in strychnia poisoning is due only to excessive strain, as we have much proof to show.

From the above results it seems evident that the toxic influence of carbolic acid in the production of convulsions and on reflex phenomena, is entirely centric. Stone (*loc. cit.*) found that when he could induce no reflex movements by irritating a sensory nerve, irritation of the cord produced marked movement in the posterior extremities; and he therefore concludes that the spinal action is probably confined to the motor columns. If this is so, the phenomenon of a consentaneous existence of exalted sensibility and motor depression is readily explainable in the

[1] See Case XLVIII. for similar symptoms in man.

fact that both conditions are due to paralysis; the exalted sensibility to a paralysis of reflex inhibitory centres,[1] and the motor depression to a paresis of the motor columns of the cord.

The *cerebral* functions are early and seriously affected, the first symptoms, as at times observed, being restlessness, bewilderment, hesitancy of speech, depression of spirits, and delirium, stupor, insensibility, and coma.

The action of the acid on the *circulation* has not been thoroughly studied. Hoppe-Seyler (*loc. cit.*) found that the arterial tension was not materially affected until convulsions were present, when it was decidedly increased, remaining in this condition for a prolonged period, and then gradually falling and continuing in a decline until it reached a point far below the normal. Salkowski and others found that the heart after death was arrested in diastole, and that this condition existed whether by slow or rapid poisoning. In man the heart generally presents strong evidence of the result of a decided and direct poison, and is to be found in a pale, flabby, and distended condition; although in several instances the left side especially was found to contain but little blood, and in at least one case this viscus was described as being healthy and empty. The increase of arterial pressure as observed by Hoppe-Seyler would seem at first sight to be dependent upon the convulsions, but Lemaire's and Salskowski's experiments seem indicative that there are other features present, for the former observer states that the systoles in the early part of the poisoning are increased in their power, and that the arterioles can be seen to contract; Salkowski declares that the rapidity of the blood-current in the capillaries is primarily increased and afterwards diminished. While there can be no doubt that carbolic acid acts ultimately as a direct and decided cardiac depressant and causes death very frequently, if not in the large majority of cases, in this way, yet the results of the above experiments are such as to preclude any decisive opinion until a further research is made. The frequent, feeble, and intermittent pulse, so frequently noticed in man, is strong evidence of a decided depressant action on the heart, as is also the slow and feeble pulse, which, at times, has been noted.[2]

The *respirations* in the earlier stages of the poisoning are decidedly increased in frequency, but later they are deep, slow, and laborious, and possess a marked stertorous character. According to Salkowski, the frequent respirations are very shallow and are almost entirely costal in their character, the diaphragm taking but little part. It was also found that respirations were accelerated in animals with cut pneumogastric nerves; and, furthermore, in normal animals, when the vagi nerves were cut dur-

[1] For a discussion of "Convulsions due to Depression of Spinal Reflex-Inhibitory Centres," etc., see my paper in *Phila. Med. Times*, Aug. 13, 1881, p. 711.

[2] Several experiments which I recently performed in connection with the blood-pressure, indicate that large doses of the acid, intravenously injected, cause an immediate cardiac paralysis.

ing the existence of frequent respiration, the movements became decidedly slowed, and were deep and regular. These results indicate both a centric and peripheral action, and that the altered respirations are due to a stimulation of both the pneumogastric peripheries and centres, while it is probable that the diminished respiratory movements, which occur later in the poisoning, are due to a paralysis of the same.

The *pupils* in the above case were more often contracted, but no experiments have been made on animals to determine how this action was brought about.

The *temperature* change in man is variable, as we have already seen, and in animals it has been stated to be diminished; according to Erls (*Schmidt's Jahr.* Bd. clxiv. p. 148), the acid diminishes the heat in animals in which fever was produced by injecting putrid substances. Latrobe and others found that carbolic acid causes changes in the blood-corpuscles out of the body; although no change was found to occur in the blood in the economy.

The *local action* of the poison is that of a decided irritant and caustic, for when applied to the living tissues it induces swelling, redness, and, generally, severe burning pain, which is followed by a white eschar and a loss of sensibility in the part. When applied to the skin the eschar becomes dried and translucent, presenting the appearance of being cornified. The same change has been observed by Taylor (*Guy's Hospital Rep.* 1868, xiii. p. 233) to occur in the mucous membrane of the stomach. This gentleman states that the superficial layer of the stomach mucous membrane was destroyed, and the dead part showed all the structures perfect, and they were rendered sharp in outline by a sort of cornification which had taken place, and that no inflammatory process existed. The loss of sensibility is not confined to the circumscribed tissue directly destroyed by the acid, but extends to at least an appreciable depth.

When swallowed, the mouth, throat, and œsophagus are whitened and present the appearance of being covered with a whitish, grayish, or brownish pseudo-membranous substance, which is soft or pulpy or diphtheritic, and easily detached from the underlying structures, which are then found to be in various stages of congestion, and frequently described as having a chocolate colour. The œsophageal mucous membrane is sometimes spoken of as being thickened, hardened, rigid, and corrugated.

The *gastric mucous membrane* is seldom affected throughout, and as might be expected the most formidable lesions are to be found in those portions which the acid would reach in its most concentrated condition; consequently, the cardiac end and the greater curvature are the most to suffer.

The stomach walls may so far be disorganized, that portions of the muscular coats are reduced with the mucous membrane to a pulp. The latter is generally found to be corrugated, congested, and corroded, and

covered with a pulpy or diphtheritic substance which may be of a whitish, grayish, or chocolate colour. The membrane is sometimes described as being thickened, tougher, more rigid, blackened, sodden, or chocolate-coloured. The summits of the rugæ have been observed to be whitened, while the interstices were red, and contained granular detritus; by another writer, elevated points about the size of shot, without any very marked congestion, were reported; and by still another, red-based patches, with well-defined borders, limited more particularly to the rugæ, were noted. The microscopical appearances of the mucous membrane have already been quoted from Taylor's paper (*loc. cit.*).

The *stomach* may be contracted or dilated, or be found in an intermediary condition. The amount of fluid it contains is variable, as is also its nature, for it may be of a whitish colour, or pinkish, brownish, or chocolate-coloured; may vary greatly in its consistency; and may possess in a greater or less degree the odour of the acid.

The occurrence of nausea, retching, and emesis, or the utter impossibility of inducing these phenomena by giving emetics by the stomach, seems readily and satisfactorily explained when we consider the irritant and corrosive properties of the acid; and at the same time that emesis may follow a local application of the acid to an absorbent surface. When the acid first reaches the gastric mucous membrane it at once irritates the peripheries of the vagi nerves, and, whether we have vomiting following the irritation, or whether the irritation is too quickly superseded by a physiological destruction of the nerve peripheries, is obviously determined by the relation existing between the rapidity with which the irritant effects are followed by those of a caustic character. That the peripheries of the pneumogastric nerves are soon seriously affected and functionally destroyed, is corroborated by the fact of the great difficulty or even impossibility of inducing emesis by drugs which effect this phenomenon by a direct local irritant action. Yet emesis, or the failure of the drug to induce it, cannot always be due to a local action only, unless it is dependent upon an effort of the system to eliminate the poison through the gastro-intestinal tract, for vomiting supervened in some of the above cases after a local application of the acid to absorbent surfaces such as the skin, wounds, rectum, etc. It is, therefore, probable that the emesis is, in part at least, centric, the drug acting in small quantity as a stimulant to the vomiting centres and in larger ones as a depressant.

What becomes of carbolic acid in the system, and the condition in which it is absorbed, are interesting questions which we are unable to answer. It has been suggested, since it possesses the property of coagulating albumen, that it is absorbed in some combination. Its presence in the blood can be a matter of but little doubt, since in autopsies the odour of the acid was frequently very strong and distinctly perceptible in the different viscera. Lemaire (*loc. cit.*) believes that it is *eliminated* to a considerable extent by the lungs, for he found the odour on the breath of

dogs and rabbits very perceptible; and we have instances of the elimination of the drug by this channel in at least four of the cases of poisoning in man. The lungs are certainly not the only channel by which elimination takes place, for Hoppe-Seyler (*loc. cit.*) found it in the saliva, and Salkowski (*loc. cit.*) and numerous other observers have found it in the urine, but in what form it is eliminated is a matter of much doubt. Patrouillard (*Jour. de Pharm. et de Chem.* 1871, p. 459) states that he obtained an oily liquid by means of ether which reacted to the characteristic tests, and which he thought to be pure acid; yet Salkowski believes that it exists as an alkaline carbolate.

The *urine* in over 20 per cent. of the cases was noticed to be coloured, and in eight of them was described as being smoky or brownish; in one it was a green-black, another had a violet tint by reflected light, and another was at first an olive-green, but became black and smoky on standing. Albumen was noted as occurring in a single instance.[1] The urine may or may not smell of the acid, or have a peculiar mixed or aromatic odour, and has been noted in exceptional instances to be bloody, copious, scanty, more acid, loaded with lithates, or to present a normal appearance. The coloration may make its appearance within a couple of hours and last for several days, and at times it can only be distinctly perceived when the urine is held up to the light.

The cause of the coloration appears to be still invested in much doubt, and it has been suggested that it is the result of the destruction of a part of the carbolic acid in the system and the consequent formation of an oxidative product—for it will be remembered that when the acid and potassium permanganate are brought in contact the former is oxidized and oxalic acid results—which is eliminated by the kidneys, and this fact seems to be strengthened by the researches of Salkowski, who found, as already stated, that oxalic acid appears in the urine of animals poisoned with carbolic acid. Husemann at first believed that the hæmoglobin was the cause of the colour, but he afterwards abandoned this theory because of the urine fully clearing up, after acidifying, and then heating, and especially because the odour of the acid was given off during the former process. Husemann, besides other observers, could detect no corpuscular elements of the blood in the urine; nor was there an excess of iron present. Waldenström and Almèn (*Zeitschrift Allgem. Apoth. Verein*, Jan. 1872), in a later series of experiments with similar urine, obtained, by adding sulphuric acid and then distilling, a product smelling strongly of carbolic acid, and which responded to the calcium chloride with a blue colour; and, moreover, when the product was shaken with ether, the latter left, on evaporation, drops of the acid. Similar results were obtained by Städeler (*Ann. d. Chem. und Pharm.* Bd. lxxvii. p. 17) with the normal urine of the cow by a similar

[1] Albumen is reported as being found in a case reported by Waldenström and Almèn.

process; and these results have been corroborated by Buliginsky (*Med. Chem. Untersuch.* 1872, p. 234), and Hoppe-Seyler (*loc. cit.*). The latter observer has, however, shown that the acid does not exist in the normal urine, but that the substance thus obtained is probably a product of the decomposition of indican; a fact which seems to be strengthened by the researches of Salkowski (*Centralb. f. Med. Wissensch.* 1876, p. 820), who found in cases of ileus an increase of the substance from which the acid is formed *pari passu* with the onset of the trouble; consequently, experiments of Waldenström and Almèn do not, therefore, prove that the acid they found was eliminated by the kidneys. There can be no doubt, however, but that carbolic acid does exist in the urine in cases of poisoning, as the odour as well as the reaction to characteristic tests signify; yet there must also be some new products formed, because the carbolic acid urine when subjected to nitric acid, and afterwards potassium hydrate, and then concentrated, gives a blood-red followed by a pea-green and violet colour. Carbolic acid added to the urine outside of the body will not yield this play of colours (*Schmidt's Jahr.* Bd. clxiv. p. 144). Microscopical examinations reveal carbonaceous particles, and it is difficult to determine whether the particles have anything to do with the characteristic test as given above or not, although it seems that they are the probable cause of the coloration, and possibly the factor upon which the reaction depends. Bauman and Herte have found that the sulphates disappear from the urine very early in the poisoning.

The *kidneys* in animals, according to Neumann, are found to be in a state of fatty degeneration. This is contradicted by the results of Salkowski's researches; but Rendu and Patrouillard's case (Case IX.) adds confirmatory proof to Neumann's assertion.

The *venous system* seems to be in a state of general dilatation, and as a consequence the different viscera and glands have been found in various degrees of congestion. The *membranes of the brain* are more frequently spoken of as being congested and full of dark fluid blood, and as an exception have been found natural. The *brain* is generally described as appearing healthy. The *arachnoidean fluid* has been found increased in some cases, and the odour of the acid has been detected in the cranial cavity.

The *lungs* are often described as being congested, and especially so in the most dependent portions. The bronchi have been noted to contain mucus, which may appear bloody or as a dark-red fluid; œdema and emphysema are exceptionally recorded. In at least three instances the lungs have been found healthy; and in one, the left lung was congested and the right emphysematous. A double-pneumonia was developed in Tennent's case (Case XLIII.), which he attributes to the effects of the acid. This is in accord with the statements of Lemaire, who found pneumonia occur in animals thus poisoned.

The *blood* in a vast majority of the cases is found to be dark and fluid, and in exceptional instances to contain soft coagula, and in one was everywhere firmly coagulated. In one case it remained fluid for five days, and in another it became bright red on exposure to the air. The odour of the acid has not unfrequently been detected in the fluid.

The asphyxiated condition, as can be surmised from the results of experimentation on animals, must be due to a paralysis of the pneumogastric apparatus; for it can scarcely be dependent, to any extent, upon changes in the blood disks, because no alterations are revealed by microscopical examination, and besides the blood possesses the power of becoming arterialized when exposed to the air after death (Brabant), which shows that the hæmoglobin was not destroyed, nor its ozonizing power paralyzed. The venous condition of the blood must then be due to a failure of the pneumogastric nerves and centres to properly carry on the respiratory function, and probably in part to the congested condition of the lungs and the depression of the circulation.

There are many difficulties in the way of satisfactorily accounting for the fluid condition of the blood on mere theoretical grounds, because excessive carbonization of this tissue seriously affects its coagulating property; yet, since we are aware of the power of the acid to coagulate albumen, it may be that it exerts some destructive influence on the fibrin-forming proteids.

The smallest *dose* which proved fatal in any of the above cases was one drachm, in a man of 64, and who died in twelve hours; yet two children, one of two and the other of two and a half years, who had each taken the same quantity, recovered. The largest dose taken was by a woman of 35, who drank something less than eight ounces; a half an ounce is almost invariably fatal, yet a case has recovered after the ingestion of four ounces, and another after taking an ounce. The most frequent dose was from a half an ounce to an ounce. Death occurred in nine cases in less than an hour; in one within three minutes, and in four others in less than twenty minutes. Life may be prolonged for twenty-four to forty-eight hours, or longer; but death usually occurs within the first four hours. Death ensued in one case in twenty minutes, from the effects of an enema for ascarides. Davidson (*loc. cit.*) cites a case where seven drops caused alarming symptoms; and in another case, above quoted, five grains elicited well-marked symptoms of poisoning.

The number of fatal cases which have followed the external use of the acid in cutaneous affections, shows that its use, to any considerable extent, in these diseases is not altogether unattended with great danger. Deaths have occurred following its application in cancer of the cervix, and most alarming symptoms have supervened from its use as a lotion in necrosis, or as an injection in abscesses or piles; and we have at least one authenticated case in which death nearly occurred from the inhalation of its fumes.

The experiments of Kempster (*loc. cit.*) on animals, well illustrate the facility with which the pulmonary mucous membrane can absorb this vapour. About one-half of the cases proved fatal when the acid was drunk or used as an enema, while a large percentage recovered in which the poisoning ensued from its local or other use. Cerna (*Phil. Med. Times*, 1879, p. 592) found the minimum fatal dose in rabbits to be fourteen and one-eighth minims per pound, and in dogs eight and four-sevenths minims per pound; certainly a much higher ratio than in man.

The frightful rapidity with which *death* occurs in some cases suggests that it must have been due to shock, such as occurs after the ingestion of sulphuric acid and similar corrosive poisons; but in the other cases where dissolution does not take place for some time it is undoubtedly dependent upon a progressive paralysis of the vital centres. Taylor (*Phila. Med. Times, loc. cit.*) believes that his case probably died from the effects of a paralysis of the pneumogastric and sympathetic nerves which brought about a cessation of function of all the important organs; while others suggest that it may be due to a failure of the heart, asphyxia, or shock. Judging from the symptoms during the poisoning and results of autopsies, there can scarcely be a doubt but that death is usually and directly due to asphyxia or an arrest of the heart or both. In animals the heart is not arrested until the cessation of the respiration, unless the acid be injected into the veins or given in very large quantities; still, there can be no doubt, when the condition of the pulse during life is considered, as well as the strong evidence in the appearance of this organ after death, of the powerful paralyzing action.

The *diagnosis* of carbolic-acid poisoning in most cases should not be attended with difficulty, for the characteristic odour of the drug can generally be detected; and in conjunction with the white or brownish stains about the mouth, and with the most characteristic symptoms as already noted, would almost be conclusive evidence; and, especially so, if the peculiar discoloration of the urine be present. After death the lesions are pretty well defined, and the odour of the acid can generally be distinctly detected in the blood and viscera. The danger of confounding these cases with apoplexy, uræmic coma, and similar conditions must be borne in mind.

The *treatment* pursued was generally such as would be indicated in cases of poisoning by corrosive substances of this nature: emetics, the use of the stomach-pump, olive-oil, milk, eggs, sodium bicarbonate, magnesia, lime-water or chalk, stimulants, counter-irritants, warmth, etc. Bleeding was practised in one case, and as curious and interesting as it may be, the patient recovered. The emetics used were of but little avail, both on account of the inability or difficulty of the patient to swallow, and the disorganized condition of the gastric mucous membrane.

Considering the difficulty of introducing emetics into the stomach, and the disorganization which has taken place in its mucous membrane, it must

be certain that where we have a choice of emetics one should be selected whose action is on the vomiting centres, and which can be used hypodermically—not one which induces emesis by a local irritant action. We cannot hope for much in these cases by using zinc sulphate, mustard, or similar substances, for even if they are gotten into the stomach, the probability is that the mucous coat is so far benumbed or destroyed that it will not respond; while on the other hand, if a centric emetic, such as apomorphine, is injected hypodermically, it will be rapidly absorbed, and the chances of its causing vomiting are infinitely greater than those with the former class.

The administration of the alkalies, especially lime in the form of a saccharate, seems to be founded upon good grounds, for according to Husemann (*loc. cit.*) they possess some antidotal properties. He also found that the oil is probably of no value; yet Lemaire declares when the acid was given with it that its poisonous effects were materially modified. As it is not at all probable that there is any chemical combination whatsoever between the acid and oil, it is likely that the latter merely acts mechanically by preventing a rapid absorption of the acid. On these grounds the oil may be administered quite freely with benefit.

Up to the time of the researches of Baumann and Herte (*Zeitschrift Phys. Chem.* I.) nothing was discovered of any special value as a chemical antidote for carbolic acid, but if the researches of these gentlemen prove to be of the value they seem invested, we have invaluable antidotes in the soluble alkaline sulphates. Baumann believes that when a soluble sulphate is given a *sulpho-carbolate* is formed that is innocuous. My friend, Cerna, found in ten experiments on animals (*Philadelphia Med. Times*, 1879, i. p. 592–597) decided evidences of the efficacy of these salts in this form of poisoning. He first decided the minimum fatal dose in rabbits and dogs, and after determining this point, gave what was deemed a toxic amount of the acid in conjunction with the sodium or magnesium sulphate, and in every instance where they were thus given together the animals recovered; yet, in repeating the experiments in the same animals, after due time had elapsed for recovery, with the same amount of acid, but without the soluble sulphate, fatal results followed; thus clearly proving the antidotal properties. It was then sought to determine if a sulpho-carbolate were formed in the economy, as believed by Baumann; consequently, the acid and solutions of the sulphates were mixed together in different proportions and different strengths, but he could obtain no chemical combination, and therefore doubted if such really takes place in the economy. Yet, until we have more conclusive proof than this, the question must still remain *sub judice;* the fact, moreover, of the disappearance of the sulphates from the urine very strongly intimates that the sulphuric acid is gotten rid of in this way.

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